Derangements of Cerebral Physiology by Anesthetic Agents or Techniques

Clark A. Sammartino, D.M.D.

PART II

Hypothermia

The use of hypothermia during anesthesia also has profound consequences for cerebral circulation and metabolism. These changes can be reflected in the electroencephalogram. Studies in the changes in cerebral blood flow in man are actually very few in number, and one can only infer that similar processes occur in man as in the well studied dog.

In the dog, each fall of 1°C in esophageal temperature is associated with a 6.7 per cent reduction in cerebral circulation of the pre-cooling flow, along with a reduction in mean arterial blood pressure and in cerebral oxygen consumption.

"Cooling from 35°C to 26°C in the anesthetized state reduces cerebral oxygen consumption by about 5 ml. per 100 g. of tissue per minute, the respiration of the brain diminishing by 0.6 ml. per 100 g. of tissue per minute with each 1°C drop in esophageal temperature. The cerebral metabolic rate is actually lowered to 35% of its pre-cooling value, when an esophageal temperature of 26°C is reached. At a temperature of 20°C anesthetized dogs can survive total cerebral ischemia lasting about 15 minutes without morbid sequela being evident on rewarming."[5]

Since similar electroencephalographic changes occur in man at temperatures comparable to those in the dog experiments, electrographic monitoring can be an invaluable adjunct to the control of surgical procedures involving hypothermia and hypotension.

When humans are anaesthetized with thiopentone and then maintained on cyclopropane and O_2, and subsequently cooled to 28°C (rectal temperature), the blood flow to the brain has been found to be reduced by 60 per cent, and the consumption of cerebral O_2 to be reduced by about 65 per cent. Even though a small rise in mean arterial blood pressure occurs, there is a reduction in cerebral blood flow due to a large increase in cerebrovascular resistance of 2-3 times. The increased resistance results from a 20 per cent increase in blood viscosity during hypothermia, coupled with some degree of cerebral vasoconstriction. If shivering occurs, the blood flow to the brain falls only by 9-12 per cent as the vascular resistance rises only by 25 per cent, and cerebral oxygen consumption is then doubled. In this case, the utilization of oxygen from arterial blood by the brain is increased, as it is in the muscles, despite the reduced temperature — and a reduced cerebral blood flow.

The use of ether anesthesia in man along with an induced hypothermia to 31°C causes a fall in mean arterial blood pressure, along with an increase in cerebrovascular resistance. Here again, a reduction in cerebral blood flow occurs, but only about 25 per cent, as the cerebrovascular resistance increases by only 64 per cent. The consumption of O_2 by the brain is reduced by 50 per cent, but the utilization of glucose falls to about one-fifth of the normal value. Therefore, the cerebral respiratory quotient

is reduced from 1.0 to 0.85. A 60 per cent reduction in arteriovenous glucose difference results.

Lastly, the employment of the technique of electrographic monitoring is of particular value in cases in which the brain is subjected to periods of prolonged ischemia. This is especially true in cardiac and in intracranial vascular surgery when hypothermia is induced to permit these periods of ischemia. In patients anesthetized with nitrous oxide and oxygen and cooled to 30°C, it has been shown to be possible to maintain bilateral occlusion of the vertebral and carotid arteries for as long as 10 minutes before changes are produced in the electroencephalogram, and there are no morbid sequelae. Electroencephalographic monitoring studies indicate that young adult brains can safely tolerate complete ischemia for some 6 minutes at a 30°C body temperature, and for 8 minutes at 28°C. It must be understood that the actual ischemic safe period also varies unpredictably with the age and clinical condition of the patient.

Summary

This paper represents a review of the literature on the effects of hypercarbia, hypotension, and anesthetic management on cerebral physiology.

Carbon dioxide was found to have the most powerful influence on the cerebral circulation. The effect carbon dioxide has on cerebral blood flow depends almost entirely on its action on the cerebrovascular resistance. This action of carbon dioxide results for two reasons — first, intracranial pressure is raised by CO_2, secondly, the diameter of the cerebral vessels is decreased by carbon dioxide and active dilation of pial vessels occurs.

There is no neurogenic mechanism involved in the cerebral vasodilator response to carbon dioxide. The effect of carbon dioxide is a direct action on the smooth muscles of the vessel walls.

The use of ganglion blocking agents during anesthesia produces a reduction in blood flow. Systemic and visceral vasodilatation is induced by the use of hypotensive agents. The mean pressure in the larger cerebral arteries of the supine patient are reduced by 40-60 per cent to values of 60-30 mm. Hg. The cerebral blood flow is decreased when hypotension is induced pharmacologically due to a reduction in the pressure head driving blood through the brain.

Hypothermia has profound effects on cerebral circulation and metabolism. In the dog, a 1°C decrease in esophageal temperature is associated with a 6.7 per cent reduction in cerebral circulation, along with a reduction in mean arterial blood pressure and cerebral oxygen consumption. If an esophageal temperature of 26°C is reached, the cerebral metabolic rate is lowered to 35 per cent of its pre-cooling value. At a temperature of 20°C, anesthetized dogs can survive total cerebral ischaemia lasting about fifteen minutes without morbid sequela.

In humans induced under thiopentone and maintained on cyclopropane anesthesia and then cooled to 28°C (rectal temperature), the blood flow to the brain has been found to be reduced by 60 per cent and cerebral O_2 consumption by about 65 per cent.

The importance of electrographic monitoring in cases in which the brain is subjected to periods of prolonged ischemia, as in cardiac surgery, under hypothermia is stressed.

Bibliography

1. Bain, J. A., and Klein, J. R. (1949) "Effect of Carbon Dioxide on Brain Glucose, Lactate, Pyruvate and Phosphates." *Amer. J. Physiology,* Volume 158. P. 478.
2. Bloor, B. M., Adam, G. L., and Woodhall, B. "Direct Measurement of Intravascular Pressure in Compo-

nents of the Circle of Willis." *Arch. Surg.,* Volume 63, 1951. P. 821.
3. Brazier, M. A. B. "Physiological Effects of Carbon Dioxide on the Central Nervous System in Man." *Medicine,* Volume 22 (1951). P. 205.
4. Cooper, K., and Ross, D. *Hypothermia.* Philadelphia, 1960.
5. Evans, F. T., and Gray, C. *General Anaesthesia.* London, 1959.
6. Kayser, C. "Physiological Aspects of Hypothermia." *Annu. Rev. Physiol.,* Volume 19 (1957).
7. Kety, S. S., and Schmidt, C. F. "Effects of Altered Arterial Tensions of Carbon Dioxide and Oxygen on Cerebral Blood Flow and Cerebral Oxygen Consumption of Normal Young Men." *J. Clin. Invest.,* Volume 27 (1948). P. 484.
8. Patterson, J. L., Heyman, A., Battey, L. L., and Ferguson, R. W. "Threshold of Response of Cerebral Vessels of Man to Increase in Blood Dioxide." *J. Clin. Invest.,* Volume 34 (1955). P. 1857.
9. Pollock, G. H., Stein, S. N., and Gyarfas, K. "Central Inhibitory Effects of Carbon Dioxide." *Proc. Soc. Exp. Biol.* N. Y., Volume 70 (1949). P. 291.
10. Sokoloff, L. "The Effects of Carbon Dioxide on the Cerebral Circulation." *Anesthesiology,* Volume 21 (1960). Pp. 664-671.
11. Sweet, W. H., and Bennett, H. S. "Changes in Internal Carotid Pressure During Carotid and Jugular Occlusion and Their Clinical Significance." *J. Neurosurg.* Vol. 5 (1948). P. 178.
12. White, J. C., Verlot, M., Selverstone, B., and Beecher, H. K. "Changes in Brain Volume During Anesthesia Produced by Thiopental." *Anesthesiology,* Vol. 12 (1942). P. 308.
13. Wilson, W. P., Adam, G. L., and Schieve, J. F. "Effect of CO_2 on Cerebral Blood Flow Spinal Fluid Pressure, and Brain Volume During Pentothal Sodium Anesthesia." *Anesthesia and Analg.* Volume 32 (1953). P. 258.
14. Wolff, H. G., and Lennox, W. G. "Cerebral Circulation Effects on Pial Vessels of Variations in the O_2 and CO_2 Content of Blood." *Arch. Neurol, Psychiat.* Volume 23 (1950). P. 1097.